

Correspondence

The Editors will be pleased to receive and consider for publication correspondence containing information of interest to physicians or commenting on issues of the day. Letters ordinarily should not exceed 500 words and must be typewritten, double-spaced, and submitted in duplicate (the original typescript and one copy). Authors will be given the opportunity to review the editing of their correspondence before publication.

Organic Cognitive Impairment?

TO THE EDITOR: An epidemic of local and systemic symptoms that followed the introduction of heat-cured phenol-formaldehyde resin in an aerospace plant generated public and professional disagreements over the cause of the outbreak.¹ Even though previous psychiatric evaluations were reported to have identified "brain damage," Sparks and colleagues have rejected the suggestion of organic brain effects among affected workers based on their use of an insensitive neuropsychological assessment of cognitive functions, and they have failed to present the data from those referred for the "standard battery of neuropsychological tests." In contrast to the thoroughness with which workers were screened for psychiatric diagnoses, screening for organic cognitive impairment was limited to "Folstein's Mini-Mental State examination, a brief test of cognitive ability,"¹ which has been criticized as "insensitive" because of "high false-negative rates" from "the reliance . . . on a global estimate of cognitive status (which) obscures the presence of isolated . . . deficits."² Since the usual pattern of cognitive function in mild chronic toxic encephalopathy is measurable impairment of only one or several specific types of cognition, use of a screening or diagnostic method that does not recognize such limited impairment would certainly mean that recognizable cases would be misclassified. Even with a relatively insensitive screening tool, Sparks and co-workers identified 4 persons with scores that indicated cognitive impairment. When these 4 and 21 others with symptoms of cognitive impairment were referred for detailed neuropsychological testing, however, "none were reported to have significant cognitive deficits of recent onset." This sequence is sufficiently unexpected that a more detailed presentation of data would have been in order. When their insensitive screening method produced positive results that were uniformly false, then the method in their hands did not have the "excellent reliability and validity" that they claimed. Their hypothesis that all cases were of psychosocial origin did not explain their four cases that were screened out as having cognitive impairment.

A reasonable alternative hypothesis could be that susceptible workers could have had mild organic cognitive impairment from which some could have recovered over periods of 4 to 12 months or more. Thus, examinations while the exposures were recent could have validly identified cognitive impairment, while a screening procedure done 6 to 12 months later could have shown that many no longer had cognitive impairment, and referral tests done an additional 1 to 3 months later could have shown that all tested had recovered, if such were the case. In this scenario no one would have to be accused of being wrong, but one would have to acknowledge that the final referral neuropsychological tests did not exclude organic cognitive impairment in the symptomatic period during and immediately after the exposures.

The authors claimed an absence of evidence for neurotoxicity of low-level formaldehyde exposure, which is contra-

dicted by Kilburn and colleagues³ and by Russian experience cited by Anger and Johnson.⁴ Phenol is also neurotoxic.^{4,5} Formaldehyde and phenol could have a synergistic neurotoxic effect.

We believe we have encountered cases of neurotoxic effects of phenol-formaldehyde resins in a different setting. We have seen fewer and more sporadic cases with unprotected exposures in poorly ventilated plywood or particleboard mills, usually resulting from periods of exposure of at least several years.⁶ Our cases involved more men than women and produced objective effects on both central and peripheral nervous systems. Similar neurotoxic effects of phenol-formaldehyde resin have been reported from Russia⁷ and Egypt.⁸ We believe that Sparks and colleagues relied on an uncertain neuropsychological screening process, failed to consider the possible favorable prognosis of very mild cognitive impairment, and have underestimated the neurotoxicity of formaldehyde and phenol.

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Dr Sparks Responds

TO THE EDITOR: I appreciate the thoughts of Morton and Feldstein regarding our previous article on an outbreak of illness in aerospace workers.¹ I agree that Folstein's screen of cognitive function is relatively insensitive.² Of those who demonstrated abnormalities on the Mini-Mental State Examination, noncooperation, preexisting learning disabilities, or inattention due to depression could not be specifically addressed, as may be possible with a more detailed battery of tests combined with old school records or other data on baseline cognitive function.